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| 10/520,901 | 04/13/2005 | Toshiyoshi Fujiwara | 09857/0202272-US0 | 2780 |
| 7278 | 7590 | 08/04/2009 | EXAMINER | |
| DARBY & DARBY P.C. P.O. BOX 770 Church Street Station New York, NY 10008-0770 | | | | SHEN, WU CHENG WINSTON |
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

| | | | |
|------------------------------|------------------------|---------------------|--|
| Office Action Summary | Application No. | Applicant(s) | |
| | 10/520,901 | FUJIWARA ET AL. | |
| | Examiner | Art Unit | |
| | WU-CHENG Winston SHEN | 1632 | |

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) Responsive to communication(s) filed on 19 May 2009.
 2a) This action is FINAL. 2b) This action is non-final.
 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) Claim(s) 4-12 is/are pending in the application.
 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
 5) Claim(s) _____ is/are allowed.
 6) Claim(s) 4-12 is/are rejected.
 7) Claim(s) _____ is/are objected to.
 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) The specification is objected to by the Examiner.
 10) The drawing(s) filed on 07 January 2005 is/are: a) accepted or b) objected to by the Examiner.
 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
 a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--------------------------------------------------------------------------------------|-------------------------------------------------------------------|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413) |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | Paper No(s)/Mail Date. _____ . |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date _____. | 6) <input type="checkbox"/> Other: _____ . |

DETAILED ACTION

1. A request for continued examination (RCE) under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 05/19/2009 has been entered.

Claims 1-3 are cancelled. Claims 4, 8, 11, and 12 are amended. Claims 4-12 are pending and currently under examination.

This application 10/520,501 is a 371 of PCT/JP03/08573 filed on 07/07/2003, and claims the benefits of foreign application JAPAN 2002-198941 07/08/2002.

Claim Rejection - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

2. Claims 4-8, 11, and 12 remain rejected under 35 U.S.C. 103(a) as being unpatentable over **Morin et al.** (Morin et al., 2000, WO 00/46355, international publication date, August 10, 2000; this reference is disclosed in IDS filed on 04/25/2006, listed as reference No. BA) in view of **Li et al.** (Li et al., A hepatocellular carcinoma-specific adenovirus variant, CV890, eliminates

distant human liver tumors in combination with doxorubicin. *Cancer Res.* 61(17): 6428-36, 2001; this reference is disclosed in IDS filed on 04/25/2006, listed as reference CC).

In the reply filed on 05/19/2009, Applicant amended claims 4, 8, 11, and 12 reciting new limitation “local cancer area” and requests favorable action on the merits, which have been fully considered and they are not persuasive. Previous rejection is **maintained** for the reasons of record advanced on pages 2-4 of the Final office action mailed on 01/21/2009.

Amended claim 4 filed on 05/19/2009 reads as follows: A polynucleotide cassette comprising an hTERT promoter operably linked with an E1A gene, an IRES sequence, and an E1B gene in this order, wherein the cassette is capable of replicating in a local cancer area. Claim 5 is directed to a recombinant virus comprising the polynucleotide of claim 4. Claim 6 limits the virus of claim 5 to an adenovirus. Claim 7 is directed to an anticancer agent comprising the recombinant virus of claim 5. Claim 12 limits the recombinant virus of claim 5 by “wherein replication of the virus kills the cancer in the local cancer area”.

Amended claim 8 filed on 05/19/2009 reads as follows: A method of killing cancer cells, comprising the step of locally administering an effective amount of the recombinant virus according to claim 5 to a patient in need thereof, such that the recombinant virus is capable of replicating in a local cancer area of the patient, and wherein replication of the recombinant virus kills the cancer cell in local cancer area. Claims 9 and 10 depend from claim 8 and limit to the cancer to various recited cancers. Claim 11 is directed to a method of killing cancer cells comprising locally administering the anticancer agent of claim 7.

Morin et al., 2000 disclosed use of the hTERT promoter to selectively direct expression in cancer cells. More specifically, Morin et al., 2000 taught oncolytic viruses, in which a toxin or a genetic element essential for viral replication is placed under control of the TERT promoter. Thereby, the virus that replicates preferentially in cells expressing TERT, and thereby selectively lyses cancer cells (See *in vitro* Example 4 on transfected human cell lines, pages 35-36, and *in situ* Example 3 on transplanted human tumor 143B cells on nude mice, page 35, Morin et al., 2000).

While Morin et al. do not teach an adenovirus with IRES inserted between E1A and E1B in an adenovirus, as recited in claim 4 of instant application, operably linked to the hTERT promoter, **Li et al.** teaches an adenoviral construct comprising promoter AFP (α -Fetoprotein, a hepatocyte specific promoter) operably linked to E1A-IRES-E1B to cause efficient replication and destruction of human hepatocarcinoma cells transplanted on a mouse. Furthermore, Li et al. teaches intratumoral injection [i.e. locally administering a recombinant virus in a local cancer area as recited in the amended claims filed on 05/19/2009] of the adenoviral construct (See line 4, left column of page 6430, Li et al.).

Therefore, it would have been obvious to combine the teachings of Morin et al., with the teachings of Li et al. to arrive at the claimed vector and methods for killing cancer cells, with reasonable expectation of success by substituting AFP promoter taught by Li et al. with hTERT promoter taught by Morin et al.

It is noted that Applicant does not provide any new arguments in the reply filed on 05/19/2009. The essence of previous **Applicant's arguments** filed on 10/17/2008 is essentially

the same as that of Applicant's arguments filed 01/14/2008. The Examiner's ***Response to Applicant's arguments*** is summarized below, with revisions addressing claim amendments filed on 05/19/2009.

Applicant argues that none of the cited references disclose or suggest the hTERT promoter for driving expression of E1A-IRES-E1B construct as recited in claim 4 and from which claims 5-12 depend (See page 6 of Remark filed on 10/17/2008), and Morin does not teach or suggest, or provide any indication for the success for the combination of hTERT with any other construct, much less the E1A-IRES-E1B construct to broadly replicate in cancer cell (See page 7 of Remark filed on 10/17/2008).

In response, Morin is relied upon for teaching selective tumor cell expression using the hTERT promoter whereas Li is relied on for teachings E1A-IRES-E1B expression cassette and intratumoral administration of a recombinant adenovirus. A promoter can drive an exogenous downstream expression cassette is well known in the art and the swapping between promoters for expressing a given cassette is a common practice in the art with a reasonable expectation of success. In instant case, the claimed recombinant virus only requires an exchange of AFP promoter (α -Fetoprotein, a hepatocyte specific promoter) with a hTERT promoter (a cancer cell specific promoter) to arrive at the intended use of the viral vector in killing various cancer cells when administered intratumorally.

3. Claims 4, 5, 8, 9, and 10 remain rejected under 35 U.S.C. 103(a) as being unpatentable over **Morin et al.** (Morin et al., 2000, WO 00/46355, international publication date, August 10, 2000; this reference is disclosed in IDS filed on 04/25/2006, listed as reference No. BA) in view

of **Li et al.** (Li et al., A hepatocellular carcinoma-specific adenovirus variant, CV890, eliminates distant human liver tumors in combination with doxorubicin. *Cancer Res.* 61(17): 6428-36, 2001; this reference is disclosed in IDS filed on 04/25/2006, listed as reference CC) as applied to claims 4-8, 11 and 12 above, and further in view of **Cheng et al.** (Cheng et al., U.S. patent application No. 2003/0104625, publication date, June 5, 2003; filed Feb. 22, 2002; this reference is cited in the office action dated 06/19/2007).

In the reply filed on 05/19/2009, Applicant amended claims 4, 8, 11, and 12 reciting new limitation “local cancer area” and requests favorable action on the merits, which have been fully considered and they are not persuasive. Previous rejection is **maintained** for the reasons of record advanced on pages 4-11 of the Final office action mailed on 01/21/2009.

The teachings Morin et al. and either Li et al. have been discussed in the preceding section of the rejection of claims 4-8, 11 and 12 under 35 U.S.C. 103(a) as being unpatentable over Morin et al. in view of Li et al.

None of Morin et al. and either Li et al. teaches various cancer recited in claim 9 and osteosarcoma and brain tumor recited in claim 10 of instant application.

However, at the time of filing of instant application, treating a type of cancer cell *in vivo* using adenovirus as an anticancer agent (claims 9 and 10 of instant applicant) was known in the art. For instant, Cheng et al. teach tumor and normal tissues, including liver, kidney, lung, bone marrow, brain, spleen, and ovary, were collected from various experimental mice groups, which was administered with adenoviral vector (See paragraph [0570], Cheng et al., 2003).

Therefore, it would have been *prima facie* obvious to one having ordinary skill in the art at the time of the invention to incorporate the teachings of Cheng et al. regarding treating various

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cancer cells using adenovirus as an anticancer with the combined teachings of Morin et al. and Li et al. regarding administration of polynucleotide comprising E1A-IRES-E1B cassette expressed under the control of hTERT promoter for lysis of cancer cells to arrive at the method of killing brain cancer cells *in vitro* comprising the step of administering recombinant virus comprising polynucleotide E1A-IRES-E1B cassette expressed via the control of hTERT promoter, as recited in claims 9 and 10 of instant application.

One having ordinary skill in the art would have been motivated to incorporate the teachings of Cheng et al. regarding treating various cancer cells with adenovirus with the combined teachings of Morin et al. and Li et al. regarding administration of polynucleotide comprising E1A-IRES-E1B cassette expressed via the control of hTERT promoter for killing cancer cells because Morin et al teaches the activity of hTERT promoter is highly specific for cancer cells, which includes brain cancer cells taught by Change et al.

There would have been a reasonable expectation of success given (i) successful demonstration of expression of E1A-IRES-E1B cassette under both transcriptional control of human TERT promoter, by the teachings of Morin et al, and translational control, by the teachings of Li et al for killing cancer cells via intratumoral administration, and (ii) the demonstration of hTERT promoter control the transcription of adenovirus E4 gene by Cheng et al. (See Figure 49, Change et al.)

Thus, the claimed invention as a whole was clearly *prima facie* obvious.

It is noted that Applicant does not provide any new arguments in the reply filed on 05/19/2009. The essence of previous *Applicant's arguments* filed on 10/17/2008 is essentially

the same as that of Applicant's arguments filed 01/14/2008. *Applicant's arguments* and Examiner's *Response to Applicant's arguments* are summarized below, with revisions addressing claim amendments filed on 05/19/2009.

(i) Applicant argues that in contrast to the Examiner's conclusion relating to the combination of Li, Morin, and further in combination with Cheng, a skilled artisan would have understood that the expression of E1B gene under the control of IRES sequence would not be at a sufficient level to cause tumor cell lysis via viral replication. Applicant argues that even if a skilled artisan reading Morin decided to control the expression of E1B at the translational level, there would have been no reasonable expectation that the arrangement of IRES sequence upstream of E 1B gene would successfully control the expression of E1B gene at a level sufficient to cause tumor cell lysis by viral replication. Therefore, there would have also been no expectation of success or motivation to combine any of these elements described Li, Morin, or Cheng as alleged by the Examiner (See pages 8-10 of Remark filed on 10/17/2008).

In response, as responded in the maintained rejection of claims 4-8, 11, and 12 under 35 U.S.C. 103(a) as being unpatentable over Morin et al. in view of Li et al., Morin is relied upon for teaching selective tumor cell expression using the hTERT promoter whereas Li is relied on for teachings E1A-IRES-E1B expression cassette and intratumoral administration. It is emphasized again that Li teaches the same **E1A-IRES-E1B** expression cassette as claimed in instant application, not three separated pieces of DNA as Applicant argues. Cheng et al is relied upon for teaching specific tumors recited in claims 9 and 10. Li et al. has clearly demonstrated that E1A-IRES-E1B expression cassette under the control of AFP promoter (α -Fetoprotein, a

hepatocyte specific promoter) functions in hepatic cancer. Furthermore, Cheng et al further demonstrates that hTERT promoter controls the transcription of adenovirus E4 gene in the context of oncolytic adenoviral vectors (See abstract and Figure 49, Change et al.). The Examiner maintains the position that there would have been a reasonable expectation of success to arrive at the claimed invention given the combined teachings of Morin et al. Li et al., and Cheng et al.

Applicant's arguments pertaining to the improvements and advantages of claimed cassette, adenoviral constructs, and methods (See page 10 of Remark filed on 10/17/2008) have been fully considered and found not persuasive. It is emphasized that the intended use of the products (claims 4-7 and 12) bears limited, if any, patentable weight, and the claimed methods (claims 8-11) as written only require an active step of administering the claimed recombinant viral vector, which leads to the killing of any given cancer cell either *in vitro* or *in vivo*. The clinical data disclosed in the Declaration filed by Toshiyoshi Fujiwara on 10/17/2008 appears to be more relevant to the previous withdrawn enablement rejection. The Declaration does not provide any evidence that renders the claims as written non-obvious or could not be practiced without a reasonable expectation of success.

(ii) Applicant argues the claimed invention is non-obvious because of secondary considerations pertaining to commercial success of Telomelysin® (OBP-301), which contains the polynucleotide construct of claim 4 and is a viral construct encompassed by claim 5 (See page 11 of Remark filed on 10/17/2008).

In response, it is noted that increase in the sale of a given product in a market is determined by multiple factors, including marketing strategy, business methods for selling the

product, increase in the demand etc. There is no evidence on the record, the asserted commercial success of Telomelysin® (OBP-301) has anything to do with the asserted non-obvious of the products and methods claimed in instant application. In particular, MPEP §716.03 states that Applicants bear the burden of proof for establishing a nexus between the claimed invention and commercial success. The Federal Circuit has acknowledged that applicant bears the burden of establishing nexus, stating:

In the *ex parte* process of examining a patent application, however, the PTO lacks the means or resources to gather evidence which supports or refutes the applicant's assertion that the sale constitutes commercial success. *C.f. Ex parte Remark*, 15 USPQ2d 1498, 1503 (Bd. Pat. App. & Int. 1990) (evidentiary routine of shifting burdens in civil proceedings inappropriate in *ex parte* prosecution proceedings because examiner has no available means for adducing evidence). Consequently, the PTO must rely upon the applicant to provide hard evidence of commercial success.

In the instant case, Applicants have provided an Exhibit that shows a license agreement and a press release speculating financial terms of the agreement, but no hard evidence of commercial success pertaining to non-obvious of the products and methods claimed in instant application.

(iii) Applicant argues the claimed invention is non-obvious because of secondary considerations pertaining to failure of others and long felt unsolved need. Applicant cites two publications (Rodriguez et al., *Cancer Res.*, 57:2559- 2563, 1997; and Kurihara et al., *J. Clin. Investig.*, 106:763-771, 2000) that describe the targeting and other problems associated with adenoviral constructs. Applicant states that **Rodriguez et al.** describes adenoviral constructs selective for antigen-positive prostate cancer cells, and **Kurihara et al.** describes adenoviral constructs selective for human breast carcinoma cells expressing the MUC1 antigen. Applicant argues that while these constructs target specific cancers, they do not exhibit the broad selective

targeting for other types of tumors as demonstrated by the results using the claimed constructs
(See page 12 of Remark filed on 10/17/2008).

In response, it is unclear to the Examiner what exactly the long-felt unsolved need is met by claimed invention but failed by Rodriguez et al. and Kurihara et al. MPEP §716.04 shows that applicants must establish that a long-felt need requires objective evidence that an art recognized problem existed for a long period of time without solution by others, that it must not have been satisfied by another before the invention by applicants, and that the instant invention must satisfy the long-felt need. In particular, a long-felt need should be analyzed as of the date the problem is identified and articulated and there is evidence of efforts to solve that problem.

Cancer research has been ongoing for decades, and to the best of Examiner's knowledge on cancer research, selective killing of cancer cells without harming non-cancer cells have been the ultimate goal, i.e. the long-felt need for effective therapeutic cancer treatment. What Rodriguez et al. (replication competent adenovirus targeting prostate cancer cells) and Kurihara et al. (replication competent adenovirus targeting breast cancer cells) accomplished are steps moving forward to this goal, despite differences in targeting mechanisms. There is no evidence on the record that the asserted "broad selective targeting for other types of tumors as demonstrated by the results using the claimed constructs" is considered as long-felt unsolved need in the field of cancer research. To the best of Examiner's understanding of the status of art relevant to the claimed invention, "broad selective targeting" refers to oncolytic adenoviruses that can selectively replicate in cancer cells and lead to the lysis of cancer cells (i.e. broadly reads on lysis of any cancer cells that propagate faster than normal cells). However, oncolytic adenoviruses are well known in art at the time of filing of instant application, including Morin

et al., and Cheng et al. cited in the maintained 103 rejections in this office action. Applicant should clarify what exactly the long-felt unsolved need is met by claimed invention but failed skilled artisan in the field of cancer research. For art rejections, Applicant is reminded again that the intended use of the products (claims 4-7 and 12) bears limited, if any, patentable weight, and the claimed methods (claims 8-11) as written only require an active step of intratumoral administering the claimed recombinant viral vector, which leads to the killing a of any given cancer cell *in vivo*.

4. Claims 4-12 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Morin et al.** (Morin et al., 2000, WO 00/46355, international publication date, August 10, 2000; this reference is disclosed in IDS filed on 04/25/2006, listed as reference No. BA) in view of **Yu et al.** (US 6,692,736, issued on 02/17/2004, filed on 03/21/2001).

Amended claim 4 filed on 05/19/2009 reads as follows: A polynucleotide cassette comprising an hTERT promoter operably linked with an E1A gene, an IRES sequence, and an E1B gene in this order, wherein the cassette is capable of replicating in a local cancer area. Claim 5 is directed to a recombinant virus comprising the polynucleotide of claim 4. Claim 6 limits the recombinant virus of claim 5 to an adenovirus. Claim 7 is directed to an anticancer agent comprising the recombinant virus of claim 5. Claim 12 limits the recombinant virus of claim 5 by “wherein replication of the virus kills the cancer in the local cancer area”.

Amended claim 8 filed on 05/19/2009 reads as follows: A method of killing cancer cells, comprising the step of locally administering an effective amount of the recombinant virus

according to claim 5 to a patient in need thereof, such that the recombinant virus is capable of replicating in a local cancer area of the patient, and wherein replication of the recombinant virus kills the cancer cell in local cancer area. Claims 9 and 10 depend from claim 8 and limit the cancer to various recited cancers. Claim 11 is directed to a method of killing cancer cells comprising locally administering the anticancer agent of claim 7.

Morin et al., 2000 disclosed use of the hTERT promoter to selectively direct expression in cancer cells. More specifically, Morin et al., 2000 taught oncolytic viruses, in which a toxin or a genetic element essential for viral replication is placed under control of the TERT promoter. Thereby, the virus that replicates preferentially in cells expressing TERT, and thereby selectively lyses cancer cells (See *in vitro* Example 4 on transfected human cell lines, pages 35-36, and *in situ* Example 3 on transplanted human tumor 143B cells on nude mice, page 35, Morin et al., 2000).

While Morin et al. do not teach an adenovirus with IRES inserted between E1A and E1B in an adenovirus to be administered and replicated locally as recited in claims 4 and 8 of instant application, operably linked to the hTERT promoter, **Yu et al.** teaches cell-specific adenovirus vector comprising target cell-specific TRE (transcriptional regulatory element) operably linked to E1A-IRES-E1B and intratumoral administration of the adenoviral vector, whose replication leads destruction of xenografts of cancer cells grown in a mouse (See Figures 1and 2, lines 12-16 of column 61, lines 8-17 of column 63, Yu et al.).

With regard to cancer recited in claims 9 and 10, Yu et al. teaches hepatocellular carcinoma (HCC) cells, gonadal and other germ cell tumors (especially endodermal sinus tumors), brain tumor cells, ovarian tumor cells, acinar cell carcinoma of the pancreas, primary

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gall bladder tumor, uterine endometrial adenocarcinoma, and any metastases of the foregoing (which can occur in lung, adrenal gland, bone marrow, and/or spleen). Yu et al teaches that in some cases, metastatic disease to the liver from certain pancreatic and stomach cancers produce AFP, especially preferred as target cells for an AFP-TRE are hepatocellular carcinoma cells and any of their metastases (See bridging paragraph of columns 27-28, Yu et al.).

Therefore, it would have been *prima facie* obvious to one having ordinary skill in the art at the time of the invention to combine the teachings of Morin et al., regarding the tumor cell and tissue specificity of hTERT promoter and its transcriptional regulation in an adenovirus with the teachings of Yu et al. regarding a bicistronic E1A-IRES-E1B cassette expressed by a cell-type specific TRE (transcriptional regulatory element) to be administered intratumorally, to arrive at the claimed vector and methods for killing cancer cells.

One having ordinary skill in the art would have been motivated to combine the teachings of Morin et al and Li et al. because hTERT promoter taught by Morin et al. activate transcription in specifically in tumor cells, and IRES taught by Yu et al. in an intratumorally administered adenoviral vector controlling the expression of E1A and E1B at translational level.

There would have been a reasonable expectation of success given (i) successful identification human TERT promoter and demonstration of hTERT promoter driven reporter gene expression at transcription level by the teachings of Morin et al. and (ii) the successful construction and expression from the E1A-IRES-E1B construct, and its translational regulation of E1A and E1B expression exerted by IRES, and intratumoral administration of the adenoviral construct, by the teachings of Yu et al.

Thus, the claimed invention as a whole was clearly *prima facie* obvious.

Conclusion

5. No claim is allowed.

Applicant is reminded that upon the cancellation of claims to a non-elected invention, the inventorship must be amended in compliance with 37 CFR 1.48(b) if one or more of the currently named inventors is no longer an inventor of at least one claim remaining in the application. Any amendment of inventorship must be accompanied by a request under 37 CFR 1.48(b) and by the fee required under 37 CFR 1.17(i).

Any inquiry concerning this communication from the examiner should be directed to Wu-Cheng Winston Shen whose telephone number is (571) 272-3157 and Fax number is 571-273-3157. The examiner can normally be reached on Monday through Friday from 8:00 AM to 4:30 PM. If attempts to reach the examiner by telephone are unsuccessful, the supervisory patent examiner, Peter Paras, can be reached on (571) 272-4517. The fax number for TC 1600 is (571) 273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you

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would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Wu-Cheng Winston Shen/

Patent Examiner

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